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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Paper No. 20040329

Application Number: 09/833,740
Filing Date: April 13, 2001
Appellant(s): DRUCKER ET AL.

Stephan A. Bent
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed 30 April 2004.

(1) Real Party in Interest

A statement identifying the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

A statement indicating there are no related appeals and interferences which will directly affect or be directly affected by or have a bearing on the decision in the pending appeal is contained in the brief.

(3) Status of Claims

The statement of the status of the claims contained in the brief is correct.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is correct in part. The proposed amendment filed 30 April 2004 has been entered. The pending claims on appeal are as amended on 30 April 2004.

(5) Summary of Invention

The summary of invention contained in the brief is correct.

(6) Issues

The appellant's statement of the issues in the brief is incorrect. Issue V of the brief, whether claims 1 and 9-11 include new matter, has been obviated by the amendment filed 30 April 2004.

(7) Grouping of Claims

Appellant's brief includes a statement that claims 1-5, claim 9, claim 10, and claim 11 do not stand or fall together and provides reasons as set forth in 37 CFR 1.192(c)(7) and (c)(8). The rejection of claims 1-5 stand or fall together because appellant's brief does not include a

statement that this grouping of claims does not stand or fall together and reasons in support thereof. See 37 CFR 1.192(c)(7).

(8) Claims Appealed

The copy of the appealed claims contained in Appendix C to the brief is correct.

(9) Prior Art of Record

No prior art is relied upon by the examiner in the rejection of the claims under appeal.

(10) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

Claims 1-5 and 9-11 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. .

Claims 1-5 and 9-11 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1-5, 9, 10 and 11 are broadly directed to a recombinant DNA construct comprising "a promoter region of a GLP-2 receptor gene", (GLP-2R promoter), which comprises at least the last 1000 nucleotides upstream of the start site of transcription of either the murine nucleotide sequence of SEQ ID NO: 1 (isolated from mouse) or a mammalian homolog of the nucleotide sequence of SEQ ID NO: 1. The specification (para. 0043) describes the promoter as comprising at least 1000 bases upstream of the transcription start site, and including at least "the number of bases necessary to drive transcription at levels above detectable background", and

desirably "transcription factor binding sites and upstream activator sequences, through which expression from the endogenous GLP-2R gene normally is regulated." The specification discloses a sequence of about 1.5 kb from upstream of the transcription start site (a SmaI-PstI fragment, nucleotides 182-1673 of SEQ ID NO: 1) of the mouse GLP-2R gene as being one embodiment. This is the only disclosed sequence that comprises at least 1000 bases upstream of the transcription start site of a mammalian GLP-2R gene. The specification discloses that the promoter of GLP-2R genes from other species, such as livestock and poultry, and mammals, especially human, are part of the invention, as are variants of these sequences, which may include truncations, extensions and deletions, but "which retain GLP-2R promoter function as determined by any of the assays herein described." The only assay disclosed in the specification relating to promoter function is to operably link the putative promoter sequence to a reporter gene, e.g. *lacZ*, make a transgenic mouse containing the construct and then compare the expression of the reporter to the expression of the endogenous mouse GLP-2R receptor in various tissues. However, the specification does not disclose what results of this assay would indicate that the putative promoter sequence is a "promoter of a GLP-2 receptor gene" required by the claimed invention. The specification at para. 0123 and 0124 discloses that the 1.5 kb mouse sequence directed expression of the reporter in similar but not identical tissues as the endogenous GLP-2R gene. Specifically, differences observed were that the endogenous gene, but not the reporter, was expressed in pituitary; the reporter, but not the endogenous gene, was expressed in lung. Also, para. 0156 discloses that the number of cells in hippocampus and cerebellum expressing the endogenous gene and reporter gene were different. The specification discloses (para. 0156) that the 1.5 kb fragment may not have all of the DNA regulatory

sequences required for correctly specifying "transgene transcription in all cells and tissues expressing the endogenous GLP-2R receptor."

Therefore, specification and claims do not clearly set forth the metes and bounds of "promoter region of a GLP-2 receptor gene" because it does not indicate what level of similarity in structure and in tissue expression between the putative promoter sequence and the endogenous promoter are required for the putative promoter to be considered a "promoter region of a GLP-2 receptor gene." It is unclear, especially from the teachings in para. 0156, whether the 1.5 kb mouse GLP-2R DNA fragment is a "promoter region of a GLP-2 receptor gene" since it did not "correctly specify transcription in all cells and tissues expressing the endogenous GLP-2R receptor." It is unclear how dissimilar a putative promoter sequence can be to an endogenous GLP-2R promoter in terms of both structure and function, and still be a "promoter region of a GLP-2 receptor gene" required by the claims. Consequently, the claims do not meet the requirements of §112, 2nd para.

The court and the Board have repeatedly held (*Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016 (CA FC, 1991); *Fiers v. Revel*, 25 USPQ2d 1601 (CA FC 1993); *Fiddes v. Baird*, 30 USPQ2d 1481 (BPAI 1993) and *Regents of the Univ. Calif. v. Eli Lilly & Co.*, 43 USPQ2d 1398 (CA FC, 1997)) that an adequate written description of a nucleic acid requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it, irrespective of the complexity or simplicity of the method; what is required is a description of the nucleic acid itself. It is not sufficient to define DNA solely by its principal biological property, because disclosure of no more than that, as in the instant case, is simply a wish to know the identity of any DNA with that biological property. Naming a type of material

generically known to exist, in this case a "promoter region of a GLP-2 receptor gene," in the absence of knowledge as to what that material consists of, is not a description of that material. When one is unable to envision the detailed constitution of a complex chemical compound having a particular function, such as a nucleic acid, so as to distinguish it from other materials, as well as a method for obtaining it, conception has not been achieved until reduction to practice has occurred, i.e., until after the nucleic acid has been isolated. Thus, claiming all DNA's that achieve a result without defining what means will do so is not in compliance with the description requirement. Rather, it is an attempt to preempt the future before it has arrived.

The instant specification discloses at most a single species readable on a "promoter region of a GLP-2 receptor gene," the region upstream of the transcription start site in the mouse GLP-2R gene sequence shown in Fig. 1a-1b. However, as indicated above, it is unclear whether the 1.5 kb mouse GLP-2R DNA fragment from this region is a "promoter region of a GLP-2 receptor gene" required by the claims. If, as suggested in para. 0156, additional DNA regulatory sequences from the genome are required "to correctly specify transgene transcription in all cells and tissues expressing the endogenous GLP-2R receptor," the specification does not describe these additional sequences. The specification discloses approximately 200 bases upstream of the transcription start site of the human GLP-2R gene, which was approximately 70% identical to the corresponding region of the mouse gene, and that the sequence upstream of this diverged. The upstream human sequence was not disclosed. However, as these two sequences are aligned in Fig. 7b, the sequence identity between the human and mouse sequences upstream of the transcription start site is only 54% (relative to the human sequence of 210 nucleotides). A best fit alignment performed by the Office of nucleotides 1-210 of SEQ ID NO: 7 (human) to

nucleotides 1474-1665 of SEQ ID NO: 1 (mouse) yielded sequence identity of 67.5%. No structural information for the promoter of a GLP-2R gene for any other species of organism is disclosed. In the disclosed mouse (SEQ ID NO: 6) and human (SEQ ID NO: 7) sequences, several potential transcription factor recognition sequences are identified (Fig. 7b) for CdxA, GATA-1, NF-κB and Sp1, all of which occur within the first 180 nucleotides of the transcription start site. No information is presented as to whether these "putative" recognition sequences are in fact utilized in transcription, much less whether they are necessary or sufficient "to correctly specify transgene transcription." Also, no information is provided as to what other sequences upstream of the first 200 nucleotides are required "to specify correct transgene transcription" relative to an endogenous GLP-2R promoter. The specification does not identify those sequences in any GLP-2R promoter, including from mouse, that are necessary and sufficient to provide for correct function of a "promoter region of a GLP-2 receptor gene" Consequently, the specification fails to identify any structural characteristics that would distinguish a "promoter region of a GLP-2 receptor gene" from other DNA sequences or promoters, i.e. those structural features of the recited promoter region that confer correct function.

While the specification provides characterization of the basal tissue expression profile of the endogenous mouse GLP-2R promoter (and the 1.5 kb fragment) in a mouse under normal laboratory conditions, it does not provide information on other factors that influence transcription, such as response to environmental conditions which alter the normal expression of the GLP-2R gene, e.g. response to inducers or suppressors of expression. Furthermore, it is unclear how the function of GLP-2R promoters from other organisms than mouse is to be assessed, in making a transgenic mouse or in making a transgenic organism of the organism from

which the promoter was obtained, e.g. making a transgenic human to assess the function of a putative human GLP-2R promoter. The specification does not disclose assays using non-mouse transgenic organisms, nor does it provide tissue expression information on endogenous GLP-2R promoters in non-mouse organisms, e.g. human, dog, sheep, cow, elephant, etc. The specification does not disclose which organisms even have a GLP-2R gene other than human, rats, and mice, although one presumes such a gene is present in other mammals.

Therefore, the specification does not provide an adequate description of a generic "promoter region of a GLP-2 receptor gene," either in terms of structure or function, and it is unclear whether it describes even a single species adequately, i.e. the mouse GLP-2R promoter, such that one of skill in the art would recognize that Applicant had possession of the invention as it is broadly claimed, or that one could envision the recited GLP-2R promoter region from the description in the specification. The rejections under §112, 1st and 2nd paragraphs could be overcome by limiting the promoter in the claimed DNA construct to "a promoter region of a GLP-2 receptor gene" with --a promoter comprising nucleotides 182-1673 of SEQ ID NO: 1--, which corresponds to the 1.5 kb fragment of mouse DNA used in the examples.

(11) Response to Argument

Response to arguments in sections 8.II.A., 8.III.A, 8.IV.A. and 8.V.A. of the brief

Appellant points out that the specification discloses partial structures of the promoter regions of the murine and human GLP-2 receptor (GLP-2R) genes, and discloses where the promoter region lies within the endogenous mouse, rat, and human GLP-2R genes (brief, pages 12-13). It is noted that the term "murine" embraces all rodents, not just house mouse; SEQ ID NO: 1 is house mouse genomic sequence. Appellant asserts that disclosure of the location of the

promoter region as being upstream of the start site of transcription conveys a correlation between the physical property of the claimed tissue-specific promoter regions and their function. Appellant concludes that the written description requirement is satisfied by these disclosures.

In response, Appellant admits in the brief that only a partial sequence of the promoter regions for the promoter region of the mouse and human GLP-2R genes is provided. The specification indicates (para. 0156) that the 1.5 kb region of SEQ ID NO: 1 does not contain all the elements present in the promoter region of the mouse GLP-2R gene. Significantly less of the promoter region of the human GLP-2R gene is disclosed. No part of the promoter region of any other mammal is disclosed. While the specification shows that the 1.5 kb region of SEQ ID NO: 1 has promoter function, albeit somewhat different from that of the endogenous promoter region of the mouse GLP-2R gene, it does not disclose that the 200 nucleotide partial sequence from the promoter region of the human GLP-2R gene has promoter function. In short, Appellant admits they were not in possession of a complete promoter region of a GLP-2R gene from mouse or human or any other mammal.

No explanation is provided as to how the location of the promoter region within an endogenous GLP-2R gene relates to tissue-specific promoter function. In general, the promoter regions of all mammalian genes include sequences upstream of the start site of transcription. The promoter regions of some genes include sequences downstream of the transcription start site, but according to the specification that is not the case for GLP-2R genes. Knowing that the promoter region of a mammalian GLP-2R gene is upstream of the transcription start site tells one where to find it in a process for making the promoter region, and where to put it in making the claimed constructs. However, it does not tell one what it is, what elements of it are necessary and

sufficient to confer the specific function of the promoter region of a mammalian GLP-2R gene, or how the structure of the promoter region itself relates to the tissue-specific promoter function of the promoter region.

Appellant also argues (brief. pp. 13-14) that the promoter regions of mammalian GLP-2R genes have homology because the first 200 nucleotides upstream of the transcription start sites of the human and mouse GLP-2R genes have homology and the sequences downstream of the transcription start sites of the mouse, human and rat GLP-2R genes have homology; concluding that one of skill in the art would expect homology in the promoter regions of GLP-2R genes. Appellant then asserts that this homology conveys possession of the full scope of the claimed invention.

In response, the cloned sequence from the mouse GLP-2R promoter region was incomplete, by admission in the specification and brief. The human GLP-2R promoter region shared homology with that of mouse in the first 200 nucleotides upstream of the transcription start site, but not farther upstream (spec. para. 0104). Taken together, these results suggest that while the mouse and human GLP-2R promoter regions may share some functional elements (those in the 200 nucleotides upstream of the transcription start site), they do not share functional elements in the remainder of the promoter region. It is unclear how these results would convey to one of skill in the art that Appellant was in possession of the invention as broadly claimed, particularly in absence of evidence that Appellant was in possession of even a single species of a promoter region of a GLP-2R gene combined with evidence that the promoter regions of different mammals diverge over most of the putative promoter region. Furthermore, the specification only contains a partial description of the function of a GLP-2R promoter region

only for mouse by disclosing the types of cells and tissues in which the GLP-2R gene is expressed in the mouse. The specification does not disclose whether the promoter region of the human GLP-2R gene, for example, directs GLP-2R in the same cells and tissues in human as does the mouse promoter in mouse. Consequently, it is not known whether the functions of the mouse and human GLP-2R promoters are the same, and the significance or importance of the shared homology between the human and mouse GLP-2R promoter regions is likewise unknown.

Appellant compares the facts of the instant application to those of the *Lilly* application (pp. 14-16), and argues that because the facts are different, the claimed invention is adequately described. One fact that differs between the instant application and that of the *Lilly* application is the *Lilly* application at least disclosed the complete structure of one species of the claimed genus. The instant application, as disclosed in the application and admitted in the brief, does not disclose the complete structure of even one species of promoter region of a GLP-2R gene. Secondly, the function of the promoter regions of GLP-2R genes of different mammals has not been shown to be the same. While the specification discloses the various cells types and tissues in which the promoter region of the mouse gene is active, it does not disclose such information for any other mammal. Consequently, the specification does not even disclose the function for the promoter regions of the GLP-2R genes of other mammals. Since the promoter regions are likely to extend farther upstream than 1.5 kb of the transcription start site, and only the last 200 nucleotides of the promoter regions from mouse and human have recognizable homology, one can conclude that either the human and mouse promoter regions do not have the same function with respect to the cell types and tissue in which they direct expression, or if they have the same function in this respect different structures are responsible for that function. As indicated in the

rejection, and not disputed by Appellant, the specification fails to identify any structural elements of the promoter region of any GLP-2R gene that confers its specific promoter function, and fails to disclose the extent of the promoter region of any GLP-2R gene required for that function. So while the specification provides partial structure for a second species embraced by the claims, it is unclear from the disclosure whether or to what extent this homologous region is important in defining function of a promoter region of a GLP-2R gene. It is also unclear just what the specific function of the promoter region of the human GLP-2R gene is, since the specification fails to disclose that information.

With respect to the holding in *Enzo* that "the written description requirement does not demand the disclosure of a nucleotide sequence for all claims to genetic material" (brief, bottom of p. 14), the relevance of this statement is unclear. *Enzo* held that the written description requirement for a claimed species of DNA could be met by biological deposit of the DNA. The issue of whether deposit of a species of DNA provided written description for a generic claim was not decided. The instant specification does not disclose the deposit any genetic material.

Appellant asserts (brief, page 15) that the specification describes three representative species, mouse, human, and rat. As discussed above, the specification does not describe even one species completely, and there is not even a partial structure for the promoter region of rat. The only rat sequence presented is downstream of the transcription start site. Knowing only where the promoter region might be found, does not inform one of what it is.

With respect to claims 9-11, Appellant admits (brief, para. bridging pp. 12-13) that the specification discloses only a partial sequence for the mouse and human GLP-2R promoter region, and explicitly states (brief, p. 19, first para.; p. 23, first para.) that the human and mouse

"promoter region includes additional nucleotide sequences." Appellant indicates that specification discloses (paras. 47 and 97) the human genomic GLP-2R genomic sequence is known. However, para. 0047 refers to published cDNA sequences; cDNAs contain sequences downstream of the transcription start site, not upstream. While para. 0097 identifies GenBank records "corresponding to" human and mouse GLP-2R genomic sequences, these GenBank records and their sequences have not been made of record, and more importantly are not incorporated by reference. Even if the GenBank records had been incorporated by reference, such incorporation would be improper, see MPEP 609.01(p), part I.A. Consequently, the contents of these GenBank records are not part of the written description. As discussed above, knowing where the promoter region of a gene is located does not inform one of the structure/function relationship of the promoter region itself. It remains that the specification provides only a partial structure of the recited "promoter region" and no other characteristics that would allow one to distinguish a DNA containing the partial sequence that is a complete "promoter region" from one that is not.

With respect to claim 10, Appellant indicates that the specification (para. 0082) describes a 10.6 kb promoter region, which included the 1.5 kb fragment of SEQ ID NO: 1 and an adjacent upstream mouse genomic DNA fragment of approximately 9 kb from an unspecified BAC clone. However, the specification does not disclose the nucleotide sequence of this additional 9kb of mouse genomic DNA, nor has the BAC clone been deposited. While applicant may have been in possession of this embodiment, the specification does not provide any description of the structure of the 9 kb fragment, other than its size, that would allow one to distinguish it from other DNA of similar size. Furthermore, the specification discloses that the construct was used to make

transgenic mice, but does not disclose whether these mice were characterized with respect to the function of the promoter in the construct compared to that of the endogenous GLP-2R promoter region, as had been done for the 1.5 kb fragment. Consequently, it is unknown whether or not the upstream 10.6 kb fragment of mouse genomic DNA has the same function as the endogenous GLP-2R promoter region, or whether this larger fragment, like the 1.5 kb fragment, is still lacking part of the complete GLP-2R promoter region.

Response to arguments in section 8.II.B., 8III.B, 8.IV.B. and 8.V.B. of the brief

Appellant argues in essence that recitation of "at least 1,000 nucleotides upstream of the transcription start site" will have the requisite functional requirements to be a "promoter region of a GLP-2R gene", and adequately sets forth the metes and bounds of the claims. In response, Appellant is not addressing the issue. The first question is what "promoter region of a GLP-2R gene" means. This phrase has no specific accepted meaning in the art, so one must look to the specification. However, the specification is not clear on what this phrase means either; it does not provide a clear definition of this structural element. It is unclear whether the recited phrase requires only a genomic DNA fragment having minimal promoter function, which need not function the same as the endogenous GLP-2R promoter, or requires a genomic fragment having the same tissue-specific function as the endogenous promoter, or something in between.

Recitation of "at least 1,000 nucleotides upstream of the transcription start site" indicates that the 1000 nucleotides upstream is necessary, i.e. it is a required part of a "promoter region of a GLP-2R gene", but raises the question of whether the 1000 nucleotides upstream is also sufficient, i.e. whether it contains all of a "promoter region of a GLP-2R gene". The specification teaches (para. 0043) that the promoter region comprises at least 1000 bases, at least 1200 bases

and at least 1400 bases upstream of the transcription start site, or can extend beyond 8000 bases upstream. It is unclear from this list what minimum length of genomic DNA upstream of the transcription start site is required for a fragment of genomic DNA ending at the transcription start site of the GLP-2R gene to consist of the promoter region. The specification teaches (para. 0156) that the 1.5 kb of SEQ ID NO: 1 upstream of the transcription start site has promoter function similar to the endogenous mouse GLP-2R promoter, but lacking some upstream sequences required for the same function as the endogenous promoter. In summary, it is unclear from the specification whether "promoter region of a GLP-2R gene" and "comprises at least 1000 nucleotides upstream of the transcription start site" means a DNA fragment of at least 1000 nucleotides upstream of the start site of transcription of a GLP-2R gene that can function as a promoter, or a DNA fragment from upstream of the start site of transcription that includes all the sequence elements that regulate expression of a GLP-2R gene, i.e. the complete GLP-2R promoter.

Appellant's arguments in the brief illustrate the ambiguity of the metes and bounds of "promoter region of a GLP-2R gene," and the dilemma faced by one of skill in the art in interpreting the claims. The brief (page 12, at bottom) characterizes SEQ ID NO: 1, which includes about 1500 bases upstream of the transcription start site, as a "partial structure ... for the murine GLP-2R promoter region" (emphasis added), and that it "includes at least 1.5 kb of the murine GLP-2R promoter region" (emphasis added). Both characterizations by Appellant indicate that SEQ ID NO: 1 is not or does not contain a murine GLP-2R promoter region, but is only part of the promoter region. Appellant states explicitly in reference to the 1.5 kb fragment (brief, p. 23, first para.) that "the promoter region includes additional nucleotide sequences."

Using this characterization of the 1.5 kb fragment, the DNA construct used in the working example, where a fragment of SEQ ID NO: 1 (nucleotides 182-1673) comprising more than 1000 nucleotides upstream of the transcription start site was linked to a lacZ coding sequence (spec., para. 0081), would not be embraced by claim 1. In contrast, Appellant states (brief, p. 17) that "the recited promoter regions, comprising at least 1,000 nucleotides, will have the requisite functional characteristics." This characterization implies that a fragment of genomic DNA beginning 1000 bases upstream of and ending at the transcription start site of a mammalian GLP-2R gene will meet the limitation of a "promoter region of a GLP-2R gene." Using this characterization, the DNA construct used in the working example would be embraced by claim 1 since it comprises more than the last 1000 nucleotides upstream of the start site. Thus, a single species of DNA construct is excluded from the claim by one interpretation of the claim language, and included in the claim by another interpretation of the claim language.

Only when one is informed of what fragment of genomic DNA consists of the promoter region of the GLP-2R gene, can one understand what other DNA sequences comprise the promoter region and then understand the metes and bounds of the claimed invention. Since "promoter region of a GLP-2R gene" has no specific accepted meaning in the art, and the specification neither defines this limitation nor describes what genomic DNA consists of the promoter region of a GLP-2R gene for mammals in general or from mouse or human in particular, the metes and bounds of the claims are unclear.

With respect to claims 9-11, the limitations in these claims are directed to the species of mammal from which the promoter region is obtained, the length of the upstream sequence, or its partial sequence. These limitations are not germane to the rejection because it is not clear

whether or not the limitation "promoter region of a GLP-2R gene" means that the promoter used in the construct must have the same function as the endogenous GLP-2R promoter, i.e. it is a complete GLP-2R promoter region. In the case of claims 9 and 11, Appellant admits (brief, p. 19, first para.; p. 23, first para.) that the recited human and mouse sequences are not a complete GLP-2R promoter region, and the specification fails to disclose whether the 10.6 kb mouse genomic DNA is a complete GLP-2R promoter region.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

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